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DIE-BACK OF COCOA

D. KAY

PRICE TWO SHILLINGS AND SIXPENCE

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INTRODUCTION

Dying back is a condition commonly associated with cocoa and has been reported from South and Central America, the West Indies, Africa, and Asia (Urquhart, 1955). Voelcker & West (1940) said it was to be found in nearly every country where cocoa was grown. The condition starts at or near the tips of young twigs in the periphery of the tree. The twigs dry out and the disease advances progressively downwards into the main branches and finally into the trunk. Dead leaves may remain attached to a moribund twig for a period of up to two months. Large branches, crowns of trees and even whole trees are sometimes killed when the attack is severe. Many different factors have been held to cause the condition and more than one factor may be responsible. Voelcker & West (1940) described two forms of the disease, 'dying back' and 'die-back', and Crowdy (1947) also described two forms which he named 'acute' and 'chronic' die-back. However, these authors did not give clear descriptions for differentiation in the field and in this paper all moribund cocoa wood will be considered as potential die-back.

DIE-BACK IN WEST AFRICA

The term die-back covers a wide range of conditions, which are unfortunately all too common in Nigeria and Ghana and constitute a major cause of loss to the cocoa industry. No detailed estimates have been made of the loss of cocoa resulting from die-back, probably because of difficulties in making an assessment, but Crowdy (1947) concluded that the die-back caused by the infection of mirid feeding lesions by *Calonectria rigidiuscula* (Berk. & Br.) Sacc., was one of the major problems facing the West African cocoa industry. Box (1944) calculated that 20 per cent of the cocoa crop in the Gold Coast was lost as the result of mirid damage and Hale (1953) estimated this loss at 60,000–80,000 tons annually, an increase of 5 per cent. to 10 per cent. on Box's estimate. In Nigeria, Hale (1953) estimated that 20,000 tons were lost annually by mirid damage and 'black pod'. Die-back caused by *C. rigidiuscula* following mirid attack may have been included in these estimates.

Concern over the extent of die-back in West Africa has resulted in much speculation as to cause of the disease. This review discusses the factors associated with the disease, with particular reference to mirid damage, and gives the sequence of events resulting in the die-back of cocoa. Methods of control which could be adopted are also discussed.

A. Causes of Die-back

Voelcker & West (1940) summarised the theories as to the causes of die-back and said that, in the Gold Coast, drought and mirid damage were the important factors, and that in Nigeria poor soils were an additional complication. Certain other factors, some of only local importance, are also considered in this review.

i. *Insect Damage*

Mirid feeding damage is probably the most important contributary cause of die-back in West Africa, and is considered in more detail later in this review. Feeding damage by thrips (*Heliothrips rubrocinctus*), an insect that occurs throughout West Africa (Alibert, 1951), was reported to cause die-back in Nigeria (Golding, 1941).

ii. *Water Supply*

The West African cocoa belt does not have a uniform climate but typically there are 50 inches or more of rain annually, concentrated

into a wet season. The daily relative humidity at this time is usually above 70 per cent. During the dry season (December, January and February) the relative humidity decreases following the appearance of the North Easterly Harmattan wind (Chamney, 1928). This wind must increase the transpiration rate at a time when there is already a water deficit, causing the leaves to wilt and some defoliation to occur. Die-back symptoms are particularly prevalent during this 'Harmattan' season. In the Gold Coast an unsatisfactory water supply, periods of drought, exposure to Harmattan winds and the absence of overhead shade were said to be major factors contributing to die-back (Dade, 1922, Bunting, 1927 and Paterson, 1927). Thomas (1929) suggested that large scale deforestation in the Bisa area of the Gold Coast had led to a decrease in annual rainfall and to the drying out of the soil, so that Harmattan winds became more harmful and resulted in serious die-back. Where some cocoa is grown under less than 50 inches annual rainfall in Nigeria it was suggested that one of the fundamental factors causing die-back was the water status of the soil in the dry season (Anon., 1940).

iii. *Soil Fertility*

Gibberd (1938) observed that a short life and sudden death were features of Nigerian cocoa trees and that die-back was unimportant until the trees were about 20 to 25 years old, when the symptoms occurred suddenly and extensively. He associated this sudden die-back with the exhaustion of nutrients in the surface soil, because trees older than 25 years still survived in 'village compound' cocoa, presumably having benefited from village refuse. He suggested that the most important factors causing die-back were nutrient deficiencies in cocoa growing on exhausted soils, combined with an inadequate water supply. Voelcker & West (1940) commented upon the numerous failures to establish cocoa in Nigeria on various poor soils. They pointed out that rainfall in these areas was adequate for cocoa and, as mirid damage was not sufficient to be solely responsible for the resulting die-back of the cocoa, they concluded that the soil type was an important factor.

iv. *Fungal Diseases*

Two fungi, *Calonectria rigidiuscula* and *Botryodiplodia theobromae* Pat., have been found by several workers to be closely associated with the die-back of cocoa, and their importance will be discussed in detail later in this review.

Cankers, caused primarily by *Phytophthora palmivora* Butl., may later become infected with *C. rigidiuscula* and die-back may result (Crowdy,

1947). A die-back associated with a root fungus, *Fomes lignosus* (Klotzsch), has been reported from the Belgian Congo (Staner, 1928). This has not been recorded in West Africa, although it is probable that the die-back at Tombel, in the Southern Cameroons, is associated with a root fungus. Aerial symptoms similar to die-back could result when root diseases, of mild pathogenicity, attack part of the root systems of cocoa trees and leaf defoliation by the leaf blight fungi (*Marasmius* spp.) may later result in this condition occurring on the defoliated twigs.

v. *Viruses*

Paine (1945) suggested that swollen shoot disease had been present in Ghana for many years before it was shown by Posnette (1940) to be caused by a virus. This disease causes defoliation and death of twig extremities followed by the slow dying back of the branches which simulates die-back. Severe strains were reported to kill cocoa within a period of two years (Posnette, 1946), but in both Ghana and Nigeria virulent and mild strains of the virus occurred. West (1950) reported that the branches died back very slowly when infected with mild virus isolates. In Nigeria there is evidence to suggest that virus infected trees die back more rapidly if they become infected with *C. rigidiuscula.*

B. The Association of Mirids with Die-back

i. *Mirids in West Africa*

The earliest record of mirids, or capsids, on cocoa in West and Equatorial Africa is from the Belgian Congo in 1895 (Nicol, 1948). Damage was subsequently reported from the Cameroons in 1902 (Paterson, 1923), the Gold Coast in 1908 (Dudgeon, 1910a & b) and Nigeria in 1913 (Anon., 1913). Three mirid species were reported from the Gold Coast: *Distantiella theobroma* (Dist.), *Sahlbergella singularis* Hagl., and *Helopeltis bergrothi* Reut. by Dudgeon (1910a & b) and a fourth species, *Bryocoropsis laticollis* Schum., was found later (Cotterell, 1943). These four species were eventually recorded in Nigeria (Anon., 1913; Golding, 1941 and Anon., 1950). *S. singularis* and *D. theobroma* feed extensively on branches and are recognised as major cocoa pests (Anon., 1948). *H. bergrothi* was thought to be a major pest of cocoa (Anon., 1914; 1916; 1920 & 1922), but as it rarely damages twigs and branches, it is now regarded as a minor pest (Anon., 1948; Nicol & Taylor, 1954). *B. laticollis* when attacking cocoa feeds exclusively on the pods, and is not of economic importance.

ii. *Mirid Damage*

There are five nymphal instars in the life cycle of mirids on cocoa. Both the nymphs and adults feed on cocoa by inserting their stylets into the phloem, cambium, and less frequently, the xylem tissues. Saliva is pumped down the stylets and the digested cell contents extracted.

There is a low population density of mirids on cocoa. Daily collections in the Gold Coast (Squire, 1946; Anon., 1947) and in Nigeria (Anon., 1955) showed that they have a distinct population cycle with minimum numbers occurring in the period March to July and maximum numbers between December and February. Maximum peak populations of approximately 1,000 individuals per acre, or less than three per tree, have been recorded on mature cocoa and less than 60 per acre where the populations were low. In a plot of 350 trees per acre, 1,000 individuals would make approximately 70 punctures per tree daily, whereas 60 individuals would make only 4 punctures per tree daily. These feeding punctures could be made on either the pods or the vegetative parts of the tree. Pod damage is rarely serious (Nicol, 1948) but damage to the vegetative parts may lead to die-back. When mirids are numerous in a plot damage can be very extensive. Williams (1953a) classified such damage into three categories of increasing severity:

1. 'blast'
2. 'Stagheaded' cocoa
3. 'Capsid pockets'.

The 'blast' condition is characterised by small discoloured lesions on young green shoots which later may wilt, the dead brown leaves remaining attached to the trees. 'Stagheaded' trees generally have a poor canopy with numerous crown branches devoid of leaves, and in 'capsid pockets' the trees are reduced to pole-like trunks bearing numerous lateral chupons. Severe damage, i.e. 'capsid pockets', usually occurred under heavy shade (Posnette, 1943) whilst 'stagheaded' cocoa was usually found where there was no shade. Williams (1953a) found that 'capsid pockets' were frequently initiated by shade trees falling and breaking the cocoa canopy. Chupon growth was thus encouraged, providing ideal conditions for rapid multiplication of mirids.

Damage is not uniformly distributed and severely damaged and healthy trees may occur together. In Ghana severe damage was reported from all the principal soil types on which cocoa was grown and soil differences were not considered a primary cause for this variation (Anon., 1947). Posnette (1943) found that certain trees

appeared to be 'immune' or 'susceptible' to mirid attack and that others seemed 'tolerant', but none of these selections maintained their field behaviour under laboratory conditions.

iii. *Fungi associated with Mirid Damage*

Damaged cocoa tissue provides a potential site for fungal infection and Dudgeon (1910a) in the Gold Coast noted an association of fungi with *Helopeltis* damage on pods. Mayne (1917) listed fungi associated with mirid damage on cocoa in the Belgian Congo and Dade (1922) in the Gold Coast found *B. theobromae* regularly associated with mirid damage. It is doubtful if a saprophytic fungus living on the dead tissue of these lesions would result in any further damage to the tree, but the entry of a parasitic fungus may have serious consequences. A high proportion of the mirid lesions may be invaded by fungi and in the Gold Coast Crowdy (1947) estimated that 80 per cent. were thus infected. In Nigeria, where an overall estimate was not possible because individual lesions often merged, estimates obtained from discrete lesions showed that 95 per cent. infection was common (Kay, 1957).

Owen (1956) found various fungi in occluded lesions, even in those that had been covered by as much as 3 in. of living tissue. *C. rigidiuscula* was most frequently isolated, with *B. theobromae* slightly less common. Other fungi were found, but were not considered important although together they amounted to one-fifth of all the fungi isolated from lesions. Similar results have been obtained subsequently in Ghana (Usher, 1955) and in Nigeria (Kay, 1958). *P. palmivora* has rarely been isolated from mirid lesions on cocoa in West Africa.

The high proportion of mirid lesions becoming infected with *C. rigidiuscula* does not seem to be due to a close biological relationship between insect and fungus. Usher (1955) did not find spores of *C. rigidiuscula* on the mouth parts of mirids and the fungus was only occasionally recovered from their body surfaces. The high proportion of lesions infected with *C. rigidiuscula* is probably a result of the fungus being ubiquitous on cocoa (Crowdy, 1947). Owen (1956) believed that in Ghana the fungus was selectively favoured in the colonisation of mirid lesions, but there is evidence against this in Nigeria (Kay, 1959b) where similar percentages of *C. rigidiuscula* were found in occluded mechanical lesions as were found in occluded mirid lesions.

Feeding punctures were first described in detail by Squire (1946), and Crowdy (1947) showed that subsequent development depended upon whether or not they became infected with the fungus, *C. rigidiuscula*. The first stages of the development of the lesion were not affected by the presence of the fungus. The medullary ray cambium

in the cortex collapsed and, for a short distance, the medullary rays of the xylem. The phloem and cambium were largely unaffected. In an uninfected lesion secondary wood cambium was formed, which cut off the dead cortical tissue, occluded any damage in the xylem and then behaved as normal primary cambium. The dead cortical tissue eventually fell away leaving on the surface of the lesion a network of dead phloem fibres. When the lesions were infected with *C. rigidiuscula*, the damage to the medullary ray tissue was more extensive in both phloem and xylem. The mycelium was mostly confined to unlignified tissue and was largely intercellular, although some haustoria were visible within the cells. The infected tissues died and the lesions healed by the growth of callus at the edges of the lesion, which eventually occluded an extensive area of damaged cells within the xylem. The surface of the affected area on the tree was characteristically roughened and this roughness persisted for many years. There have been no parallel observations on the effects of other fungi, in particular *B. theobromae*, in the development of mirid lesions.

When the mirids' stylets do not penetrate below the cambium, the wound cambium is formed entirely below the lesion and extrudes the infection to leave no outward sign of damage. When the stylets extend below the cambium, the fungus can attack the xylem beneath the wound cambium. The fungus is unable to compete with the rapid development of wound callus in a healthy tree and becomes occluded by healthy tissue (Crowdy, 1947). When the tree is not growing satisfactorily the fungus may develop further, killing more woody tissue and causing die-back (West, 1948; Owen, 1956).

Although various fungi were isolated from feeding lesions, Crowdy (1947) concluded that woody shoots were killed only when the lesions were infected with *C. rigidiuscula*. He found that in the laboratory uninfected mirid lesions did not kill woody shoots, whereas in the field twigs ringed by *C. rigidiuscula* infected lesions died. Unfortunately he did not equate the laboratory conditions with the field ones where one might expect greater mortality of shoots than on those kept under suitable laboratory conditions. In Nigeria it has been impossible to conclude whether infected mirid lesions were entirely responsible for the die-back that occurred, because whilst some twigs ringed with infected lesions died, numerous others remained healthy and bore fruit. It was found that die-back appeared most frequently by spreading from terminal green twigs which had been killed by feeding mirids and which had subsequently been infected with the fungus.

iv. *Die-back in the Absence of Mirid Damage*

An opportunity to examine die-back in the absence of mirid damage has been provided by the use of efficient insecticides. In

Nigeria some trees, which had been coppiced in 1954 and subsequently sprayed against mirids, were examined. These trees had been given careful cultivation including leaf mulch and fertilisers and were grown under adequate shade. The annual rainfall was in the order of 45 in. In 1958, although they had been kept virtually free of mirid attack, every tree showed some die-back. An analysis of the fungi occurring in one hundred of the dead branches showed *C. rigidiuscula* and *B. theobromae* to be the commonest, and that they occurred in proportions similar to those found in die-back on mirid damaged trees. These diseased areas were freely distributed in the fan canopy, and in logitudinal section they were similar to the other die-back examined. No wounds were found on the dead twigs to show where the fungus could have entered and it is suggested that *C. rigidiuscula* must first have acted as a saprophyte on existing dead wood and later parasitised the living tissue. Field experiments have confirmed that this sequence does occur (Kay, 1959a).

C. The Association of Fungi with Die-back

In the Gold Coast, Crowdy (1947) found *C. rigidiuscula* and *B. theobromae* associated with die-back. Some 23 per cent. of the affected twigs yielded the fungus *C. rigidiuscula*, 32 per cent. *B. theobromae* and 45 per cent. both fungi. Similar results were obtained by Owen (1956) and Usher (1957). In Nigeria similar proportions of these two fungi were found although some die-back did not yield any fungi (Kay, 1958). It was indicated that *B. theobromae* was saprophytic, whilst *C. rigidiuscula* was a wound parasite. Briton-Jones (1934) stated that *B. theobromae* had been recorded on many hosts, but not as a primary parasite, and Owen (1956) showed that the spread of *B. theobromae*, when inoculated into cocoa seedlings, was negligible even after 40 weeks. *C. rigidiuscula* has been found associated with cankers on cocoa in Ceylon and the West Indies (Petch, 1910). Cocoa canker in the Cameroons was attributed to the imperfect stage of *C. rigidiuscula* (Brick, 1908). Crowdy (1947) showed that *C. rigidiuscula* could establish itself in wounds in cocoa stem tissue of all ages, and considered the fungus a wound parasite, whilst Owen (1956) showed that *C. rigidiuscula* infecting wounds spread quite rapidly in the xylem of cocoa.

Three zones were described in longitudinal sections cut through diseased wood: an outer greyish-black area of dead wood, adjoined by a narrow claret-coloured zone that darkened on exposure, which was separated from the healthy tissue by a straw-coloured water soaked zone (Crowdy, 1947). Fungi, particularly *B. theobromae*, were isolated from the dead wood but only *C. rigidiuscula* was isolated from

the claret-coloured zone, and no fungi were isolated from the straw-coloured zone. Owen (1956) found some die-back where this zonation was not apparent and considerable diversity in the fungal isolates, but he noted that *C. rigidiuscula* and *B. theobromae* were isolated with equal regularity either separately or together. In Nigeria (Kay, 1958) the usual zonation was as described by Crowdy, but some specimens did not show these well defined zones. Where full zonation occurred, *C. rigidiuscula* was isolated from the claret zone, but when the water-soaked zone was absent, *B. theobromae* was usually isolated. In some lesions the dead wood was not distinctly delineated from the adjacent probably moribund tissue, which extended for up to 12 in. below the obviously dead wood. This zone had an overall diffuse black appearance, due to irregular streaks in the wood, and it was not easy to distinguish where moribund wood ceased and healthy wood began. No water-soaked zone was visible in this type of die-back.

Owen (1956) concluded that the only important fungus causing die-back on cocoa in Ghana was *C. rigidiuscula*. He suggested that the reason for the regular isolation of *B. theobromae* and the absence of *C. rigidiuscula* from some die-back was that *B. theobromae* generally occurred behind the *C. rigidiuscula* but that sometimes the more vigorous *B. theobromae* over-ran the *C. rigidiuscula*. If this were true, one might expect that in areas where only *B. theobromae* was found the die-back would remain static until a reappearance or a new colonisation of *C. rigidiuscula* occurred. In Nigeria it was found that *B. theobromae* can be isolated from the claret-coloured zone of diseased branches on which daily increases of dead wood had been recorded. A possible explanation of this is that *C. rigidiuscula* is not present along the whole face of the advancing die-back and *B. theobromae* which is present can then be isolated. The fundamental importance of one fungus was shown when it was found that the twigs of trees died after inoculations with *C. rigidiuscula*, although similar inoculations of *B. theobromae* did not cause this die-back. In appearance and section the dead twigs were similar to twigs that had died in the field.

D. The Formation of Die-back

It would appear that there are two forms of die-back in West Africa, a physiological die-back that occurs as the result of growth-limiting ecological factors such as drought, and the die-back disease which follows the establishment and spread of the fungus *C. rigidiuscula* within the tree. In the field it is suggested that the two forms can exist at different times in the same diseased branch. Physiological die-back occurs suddenly but there are two phases in the development

of fungal die-back, the first when the fungus invades the twig and the second when it spreads into living wood.

The fungus can enter via mirid lesions, mechanical wounds or dead wood, but it is most likely that mirid feeding lesions provide the majority of entry points. The subsequent development of die-back into healthy tissue is probably controlled by two factors, the pathogenicity of the fungus and the health of the trees. *C. rigidiuscula* has been reported as a weak parasite, in that it can be occluded in healthy cocoa tissue, and it appears that the health of the tree is the more important factor. Crowdy (1947) emphasised that only healthy cocoa could occlude the fungus, whilst West (1948) thought that occluded lesions would remain dormant unless the trees were weakened, when a rapid die-back would develop from these lesions. The two factors of drought and soil nutrient deficiencies, said by Voelcker & West (1940) to be causes of die-back, are factors that would affect the health of the tree, and would therefore be of great importance once the fungus was established. In Nigeria, Kay (1959b) has shown that the condition of the host is of considerable importance. The rate of spread of *C. rigidiuscula* inside cocoa seedlings was changed by keeping the seedlings under different environmental conditions. The spread was reduced in seedlings that were grown in full sunlight, compared with ones grown in shade, and was increased if the seedlings were kept under a water deficit or given excess water. In mature trees there was a greater spread of the fungus in those having a poor leaf canopy compared with adjacent trees having a full leaf canopy. Also, the fungus spread at a much greater rate, and a given number of inoculations caused die-back on more occasions in virus-infected trees than in virus-free trees. It is thought that when physiological die-back occurs in the absence of a fungus and severe mirid damage, this dead tissue may later be invaded by the fungus to become fungal die-back.

CONTROL

In West Africa, where die-back is largely associated with severe mirid damage, considerable control may be obtained by chemical spraying against mirids. Chemical sprays for the control of mirids were reported by Dudgeon (1910a) and Cotterell (1926) but were not widely used. The more recent successful application of synthetic organic insecticides has led to the development of the large-scale control of mirids in West Africa. Spraying gamma-B.H.C. on mirid damaged plots where die-back was extensive (Donald, 1957) resulted in the regeneration of many of the affected trees. D.D.T. and aldrin have given similar results. Although B.H.C. has recently been reported

as having some fungicidal properties these are not to be considered to be of importance in controlling die-back as the fungus lies within the wood. Control therefore appears to be indirect in that the insecticide controls mirid damage, a predisposing factor to die-back. Taylor & Wharton (1955) incorporated a fungicide into the mirid spray to test the efficiency of this double spray against the combined damage of mirids and the fungus *C. rigidiuscula*. No satisfactory method of assessing the damage due to the mirids alone, compared with mirids and fungus, was found and the results obtained were inconclusive.

A cocoa tree exerts some control over the development of die-back and measurements of increases in the amount of dead wood have shown that less than 10 per cent. of the foci of infection increase in size in any one month. This extension followed a typical pattern. The spread of die-back along internodes and unbranched or leafless branches was rapid, but spread was checked at the point where a leaf or healthy twig grew from the infected branch. Extension then occurred only on the opposite side, sometimes for as much as 4 ft., to the leaf or twig insertion below. The subsequent death of the leaf or branch checking the die-back was followed by a rapid increase of die-back down the infected branch. A healthy tree produced fresh leaves and shoots below the infection and development of the die-back was thus impeded, whilst a weakened tree did not produce sufficient growth to impede the disease. Any treatment found to encourage the production of new growth would therefore automatically exert some control over the extension of existing die-back.

In West Africa two fungi, *C. rigidiuscula* and *B. theobromae*, have usually been associated with die-back, but only *C. rigidiuscula* is considered to be important. *C. rigidiuscula* is not a primary parasite and it enters the cocoa through wounds and existing dead wood. Crowdy (1947) showed that the fungus could not compete with the growth of wound callus in a healthy tree. Thus control of the disease must be directed against this specific fungus, having a limited mode of entry and unable to compete with new cocoa tissue formation.

The elimination of this fungus in the field presents a difficult problem because it is widely distributed in cocoa, having been isolated from most parts of the cocoa tree (Usher, 1957), and also it sporulates readily on dead cocoa wood (Crowdy, 1947). The manner of dissemination of the spores is not fully understood. They may be borne in water (Usher, 1957), or by insects (Crowdy, 1947), but they are not air-borne (Obamwony, unpublished; Usher, 1957) and only a slow infiltration might be expected from an outside source. The reservoir of infection could be reduced by pruning all dead wood from the trees and then spraying the trees with a fungicide. An experiment

incorporating these treatments was carried out in Nigeria (Kay, 1959b) and the number of new foci of die-back infection was reduced on the pruned trees that had been sprayed with fungicides.

Careful harvesting and judicious pruning would reduce the number of mechanical wounds on cocoa trees, but numerous entry points for the fungus would still be provided by small cracks which develop on twigs and branches in the canopy exposed to full sunlight. Mirid feeding lesions, in green and in older wood, provide ideal entry points for the fungus and before any control of die-back can be attempted mirid populations must be reduced, if not eliminated. Mirid feeding promotes die-back first by providing numerous entry points for the fungus and secondly by causing the death of leaves and twigs, enabling die-back to become firmly established. Routine spraying of cocoa with gamma-B.H.C. can control mirid populations but existing mirid damage remains. The importance of this damage in an occluded state remains controversial.

It is possible that the most effective method of control of die-back would be to control its development within the tree rather than eliminate the fungus from the cocoa or prevent is entry into the tree. There is considerable evidence that the condition of the tree may exert control over the growth of the fungus. Crowdy (1947) suggested that old cocoa trees would succumb more readily to *C. rigidiuscula* infection than young trees and inferred that growth of the fungus was more rapid in unhealthy trees. Owen (1956) found that the fungus developed at slower rates in hosts other than cocoa and that there was a variation in the development of the fungus in different varieties of *Theobroma cacao* L., whilst Usher (unpublished) found that 'lack of shade and watering of seedlings' increased the growth of the fungus in artificial lesions. Kay (1959b) has found that the spread of the fungus is affected by the condition of the host.

SUMMARY

Dying back is a condition which occurs in nearly every country where cocoa is grown and is probably a symptom of more than one disorder. In West Africa die-back has usually been associated with mirid damage or periodic drought, although the factors of soil fertility, thrips damage and fungal disease have been of local importance. There is much similarity between the death following virus infection and die-back, and virus infected trees die back more rapidly when infected with *C. rigidiuscula.*

There is a marked association between mirid feeding damage and subsequent die-back and similar fungi have been isolated from mirid damaged tissues and from die-back. Mirid lesions can only kill woody

tissue when infected with *C. rigidiuscula* and this fungus inoculated into twigs can cause die-back. It has been stated that die-back arises when the fungus in occluded mirid lesions 'bursts out' whilst the tree was in a weakened condition, but there is no evidence to support this. In Nigeria die-back usually arises from terminal green twigs that had been killed by feeding mirids, although other factors were also recognised, and then infected with *C. rigidiuscula*. Although there is no attempt to control die-back as such, a widespread control of mirids, a major predisposing factor to die-back, is carried out in West Africa. It is suggested that control measures be developed along the following lines:

1. Eliminate, where possible, the causative factors of die-back, such as mirid damage, and fungal diseases.
2. Determine the conditions that encourage the spread of *C. rigidiuscula* within the cocoa trees.
3. Search for resistance factors in the population.

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